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FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS' ENTERED AT 12:28:32 ON

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APR 2001

L1	6 S SKG AND ARTHRITIS/AB,BI
L2	6 DUP REM L1 (0 DUPLICATES REMOVED)
L3	870 S BALB## AND ARTHRITIS/AB,BI
L4	54 S L3 AND SPONTANEOUS?/AB,BI
L5	40 S L4 AND (AUTOIMMUNE OR RHEUMATOID)/AB,BI
L6	23 DUP REM L5 (17 DUPLICATES REMOVED)
L7	21 S L5 AND JOINT#/AB,BI
L8	12 DUP REM L7 (9 DUPLICATES REMOVED)
	E SAKAGUCHI SHIMON/AU
L9	45 S E3-E5
L10	7 S L9 AND BALB?/AB,BI
L11	5 DUP REM L10 (2 DUPLICATES REMOVED)

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17 and 15	0

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USPT,PGPB,JPAB,EPAB,DWPI	17 and 15	0	<u>L8</u>
USPT,PGPB,JPAB,EPAB,DWPI	sakaguchi-s\$.in.	1641	<u>L7</u>
USPT,PGPB,JPAB,EPAB,DWPI	15 and (rheumatoid adj1 arthritis)	10	<u>L6</u>
USPT,PGPB,JPAB,EPAB,DWPI	13 and "balb/c"	115	<u>L5</u>
USPT,PGPB,JPAB,EPAB,DWPI	13 and (skg)	0	<u>L4</u>
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USPT,PGPB,JPAB,EPAB,DWPI	((435/4)!.CCLS.)	3077	<u>L2</u>
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L12: Entry 9 of 13

File: USPT

Aug 21, 1990

DOCUMENT-IDENTIFIER: US 4950741 A

TITLE: Antibody against rheumatoid arthritis specific protein

Detailed Description Paragraph Right (33):

Particularly, a purified rheumatoid arthritis specific protein is administered together with complete Freund's adjuvant to a mouse such as BALB/c mouse to effect immunization of the mouse. After completion of the immunization, the spleen is taken from the mouse. Antibody-producing cells are obtained from the spleen. These cells are fused with myeloma cell-derived P3-X63-Ag8-U1 strain (parent strain) in the presence of a fusion promoter, which strain is deficient in hypoxanthine guanine phosphoribosyl transferase (HGPRT) in the presence of a fusion promoter. As the parent strain, there have been reported a variety of strains in addition to P3-X-63-Ag8-U1. As such a parent cell, there may be mentioned, for example, P3-X63-Ag8, P3-NSI/1-Ag4-1, X63-Ag8-6.5.3, Sp2/0-Ag14, FO, S194/5XX0.BU.1, etc. Therefore, cell fusion may be attained from other combinations of such parent strains with their appropriate mouse strains than mentioned above. As a fusion promoter, there may be generally employed polyethylene glycols (PEG) having different molecular weights. Alternatively, liposomes (artificial lipid vesicles), Sendai virus (HVJ), etc. may also be used as a fusion promoter for cell fusion. In addition, there may be employed a method of electric fusion in which cell fusion is effected by applying voltage to cells without using a fusion promoter. When cell fusion is effected using P3-X63-Ag8-U1 as a parent strain and thereafter cultivation is carried out in HAT culture medium, there can be obtained selectively only fused cells comprising a normal cell having an ability of producing an antibody and a cell of P3-X63-Ag8-U1.

Detailed Description Paragraph Right (76):

0.3 mg of the purified rheumatoid arthritis specific protein as obtained in Example 1 was dissolved in 0.5 ml of Dulbeccos' phosphate buffer physiological saline solution (hereinafter often referred to as "Dulbeccos' PBS"). To the resulting solution was added an equi-volume of Freund complete adjuvant (manufactured and sold by Gibco Co., U.S.A), followed by thorough mixing to give a complete water-in-oil emulsion. The obtained emulsion was subcutaneously injected to the back of BALB/c mouse to immunize the mouse. Immunization was effected further twice at intervals of three weeks in the same manner as the above. After 10 days, 0.3 mg of the purified RASP dissolved in 0.5 ml Dulbeccos' PBS was intraperitoneally injected to boost immunization. After 3 days from the final immunization, the spleen was taken from the mouse. The spleen was subjected to filtration under pressure using a stainless steel net [100 mesh (Tyler)] to obtain a mixture of spleen cells and red blood cells. The mixture was subjected to lysis using a lysing buffer (prepared by mixing 0.16 M ammonium chloride with 0.17 M Tris buffer (pH 7.65) at a ratio of 9:1 and adding thereto an appropriate amount of hydrochloric acid to give a pH of 7.2) and was thoroughly washed with Hank's solution to remove red blood cells. The thus obtained spleen cells were used as antibody-producing cells.

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L12: Entry 2 of 13

File: USPT

Aug 8, 2000

DOCUMENT-IDENTIFIER: US 6100445 A

TITLE: Transgenic knockout mouse having functionally disrupted interleukin-1.beta.
converting enzyme geneDetailed Description Paragraph Right (20):

To identify a disease condition involving matIL-1.alpha. and/or matIL-1.beta. secretion, and thus treatable by an ICE inhibitor, an attempt is made to induce the disease condition in an animal of the invention homozygous for the ICE gene disruption. In one embodiment, the attempt to induce the disease condition involves administering a stimulus to the animal that induces the disease condition in a wild-type animal (e.g., induction of septic shock by administration of lipopolysaccharide (LPS)). In another embodiment, the attempt to induce the disease condition involves breeding an animal of the invention with another animal genetically prone to a particular disease. The animals are crossbred at least until they are homozygous for the ICE null mutation. For example, an animal of the invention can be bred with an animal prone to a particular autoimmune disease to assess the involvement of IL-1 in the pathology of the autoimmune disease and to determine whether an ICE inhibitor may be effective in treating the autoimmune disease. Examples of mice strains genetically susceptible to particular autoimmune diseases include the MRL/lpr mouse (Cohen, P. L. et al. (1991) Ann. Rev. Immunol. 9:243-269), which is a model for lupus erythematosus, and the NOD mouse (Rossini, A. A. (1985) Ann. Rev. Immunol. 3:289-320), which is a model for insulin-dependent diabetes mellitus. Non-limiting examples of other mouse strains (and their disease susceptibilities) which can be bred with the animals of the invention include: DBA/1 (collagen-induced arthritis; model for rheumatoid arthritis) (Wooley, P. H. et al. (1981) J. Exp. Med. 154:688-700), BALB/c (proteoglycan-induced arthritis and spondylitis; model for rheumatoid arthritis and ankylosing spondylitis) (Glant, T. T. et al. (1987) Arthritis Rheum. 30:201-212), PL/J (experimental autoimmune encephalomyelitis; model for multiple sclerosis) (Fritz, R. B. et al. (1983) J. Immunol. 130:191-194), NZB/KN (polyarthritis; model for rheumatoid arthritis and osteoarthritis) (Nakamura, K. et al. (1991) Arthritis Rheum. 34:171-179), C57BL (osteoarthritis; Pataki, A. et al. (1990) Agents Actions 29:201-209), STR/ORT (polyarthritis; model for rheumatoid arthritis and osteoarthritis) (Dunham, J. et al. (1990) J. Orthop. Res. 8:101-104), and Tsk/+ (systemic sclerosis; Siracusa, L. D. et al. (1993) Genomics 17:748-751). For MHC-associated disease models, offspring of the crossbreeding are selected that maintain the disease-susceptible MHC haplotype. Many mouse strains genetically susceptible to particular diseases are available from The Jackson Laboratory, Bar Harbor, Me. or other commercial or academic sources. The disease condition is then induced in the crossbred animals either spontaneously or experimentally.

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L4: Entry 9 of 9

File: USPT

Jan 27, 1981

DOCUMENT-IDENTIFIER: US 4247540 A
TITLE: Therapeutic agent

Brief Summary Paragraph Right (11):

The extract is obtained by crushing the abdomen of the ant. The crushed abdomen are then mixed with an aqueous solvent and the resulting solution filtered, purified, and adjusted to provide a concentration effective for injection into a patient. The extract containing the therapeutic agent is injected subcutaneously into the patient wherein, apparently without significant side effects, it causes remission of arthritic symptoms and appears to significantly decrease the RA factor (measure of an antibody normally associated with rheumatoid arthritis). Similar treatment will also cause remission of symptoms for asthma.

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L4: Entry 8 of 9

File: USPT

Mar 13, 1990

DOCUMENT-IDENTIFIER: US 4908387 A

TITLE: Use of beta.sub.2 antagonists in the treatment of inflammatory diseases, in particular, rheumatoid arthritis

Brief Summary Paragraph Right (3):

None of the drugs presently available for treating inflammatory disorders are known to act via the nervous system. However, researchers have recently noted an apparent contribution of the sympathetic nervous system to the proliferation of inflammatory conditions and processes. It has been reported that dogs chronically maintained on beta adrenergic agonists develop a rheumatoid arthritis like syndrome [Vyden et al. Arthritis Rheum. 14, 420, (1971)]. The inventors of the present invention, in conjunction with colleagues, have reported on studies showing that sympathectomy markedly attenuates the signs of inflammation and severity of joint injury in rats with experimentally induced arthritis and that intracerebroventricular administration of morphine, which is known to decrease sympathetic tone, decreases arthritic severity [Levine et al., J.Neurosci. 6, 3423-3429 (1986)]. The inventors have also reported that regional sympathetic blockade with guanethidine reduces pain and increases pinch strength in patients with active rheumatoid arthritis [Levine et al., J. Rheumatol., 13, 1040-1043 (1986)]. Finally, propranolol, a beta adrenergic blocker, has been shown, in very high doses that produce significant toxicity, to decrease signs and symptoms of inflammation, in patients with active rheumatoid arthritis; suppression of joint deterioration was not shown. [Kaplan et al., Arthritis Rheum. 23, 253-255 (1980)].

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L4: Entry 3 of 9

File: USPT

Dec 15, 1998

DOCUMENT-IDENTIFIER: US 5849336 A

TITLE: Method using sturgeon notochord for alleviating the symptoms of arthritis

CLAIMS:

13. A method for treating rheumatoid arthritis in a human in need of such treatment comprising oral administration to said human of an amount of a component selected from notochord, extracts of notochord and mixtures thereof, said amount being effective to decrease at least one arthritis symptom.

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L4: Entry 8 of 9

File: USPT

Mar 13, 1990

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BALB/C same rheumat\$	13

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<u>L9</u>	L5 near5 developing L2	0	<u>L9</u>
<u>L8</u>	L5 near5 L2	0	<u>L8</u>
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<u>L3</u>	treatment and L2	10292	<u>L3</u>
<u>L2</u>	rheumatoid arthritis	10921	<u>L2</u>
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result set

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<u>L3</u>	treatment and L2	10292	<u>L3</u>
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